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The impact of maternal consumption of cafeteria diet on reproductive function in the offspring

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HIGHLIGHTS

- Cafeteria diet after weaning induces obesity in adulthood.
- Maternal obesity induces changes on reproductive function of male offspring.
- Male sex hormones are altered by maternal consumption of cafeteria diet.
- Maternal consumption of cafeteria diet alters sexual behavior of male offspring.

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ABSTRACT

Maternal obesity is a risk factor for the development of metabolic syndrome and childhood obesity, and early overnutrition seems to induce the development of pathologies in adulthood, including insulin resistance, cardiovascular diseases, type 2 diabetes mellitus, and a higher BMI. In addition, it is known that obesity can negatively affect fertility and reproductive function in men. The objective of this work was to investigate the impact of maternal obesity induced by the consumption of cafeteria diet on metabolic, endocrine and reproductive outcomes in the male offspring. Body weight, abdominal fat content and concentrations of insulin, leptin, glucose and total cholesterol were analyzed in dams. The same parameters were evaluated in pups when in adulthood, in addition to the analysis of sexual behavior, followed by measurement of plasma luteinizing hormone, follicle-stimulating hormone, testosterone, and prolactin. Maternal consumption of cafeteria diet affected reproductive hormone regulation in the offspring and such modifications were reflected on sexual performance. Also, these modifications were independent of time and of the reproductive period during which dams consumed the diet. Our results indicate, for the first time, that maternal nutrition may have a deep impact on the reproductive function of the adult male offspring.

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1. Introduction

Overweight and obesity are a major public health problem, which has achieved epidemic features [1,2], affecting different age groups [3,4] as a result, mainly, of changes in lifestyle, such as increased food intake and infrequent physical activity [1,5]. The incidence of obesity during pregnancy increased from 70 to 100% in the past decade, which explains almost all complications during pregnancy and also the increase in fetal health disorders, both in the short and the long run, such as congenital anomalies, birth weight and size inappropriate for gestational age, and higher risk for the development of obesity and metabolic syndrome in those born to obese mothers [6–12]. This body of evidence demonstrates that the effect of maternal obesity is passed on to the next generations, confirmed both by human and animal studies, suggesting that maternal environment during the perigestational period may trigger the development of obesity and of metabolic syndrome in the offspring during childhood and adulthood [13–19]. However, the mechanisms whereby maternal obesity may affect the health of the offspring are yet unclear.

It is also unclear whether maternal obesity may also affect different aspects of reproductive health in the offspring, such as the development and function of the hypothalamic–pituitary–gonadal axis. Several studies have shown a strong association between obesity and reproductive disorders in men, [20–22] and women [23–25], and in animal models as well [26–28]. In men, there is compelling evidence that obesity reduces the quality of spermatozoa, modifying the physical and molecular

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structure of germ cells and also of mature spermatozoa, originating from changes in male reproductive hormone levels [20,27]. In obese men, the concentrations of sex hormone-binding globulin (SHBG) are lower [22], there is a decrease in circulating testosterone, reduction in LH pulse amplitude, and hyperestrogenemia [21]. Together, these factors have been related to fertility disorders often observed in obese individuals [29]. Nevertheless, there is a paucity of studies that associate obesity with behavioral aspects of male reproduction. In addition, whether maternal obesity could be one of the causative factors of reproductive disorders in the male offspring has been underinvestigated.

Given that maternal obesity is a risk factor for the development of obesity and metabolic syndrome in childhood and that these changes often persist into adulthood, the aim of the present study was to investigate the impact of maternal obesity induced by the cafeteria diet on the metabolic and reproductive outcomes of the male offspring.

2. Materials and methods

2.1. Animals and feeding procedures

The experimental protocol was approved by the Research Ethics Committee and Animal Care and Use Committee of Universidade Federal do Rio Grande do Sul (protocol no. 2008221). All animals were housed in plastic cages (five rats/cage) and kept in vivaria under controlled temperature (20–24 °C) and lighting (12 h light–12 h dark cycle). The animals were housed on shavings and had *ad libitum* access to standard rat chow and water at all times. A total of 50 female Wistar rats aged 21 d were randomly divided into two treatment groups: (1) control group (CON), fed standard rodent chow and water *ad libitum*; and (2) cafeteria group (CAF), fed cafeteria diet adapted from previous studies [30–32]. Cafeteria diet is a selection of highly energetic and palatable human foods. The cafeteria diet included biscuits, ham, cake, marshmallow, sausage, salami, bread, snacks, gumdrop, wafer, candy and soft drink, as shown in Table 1. Excessive amounts of food plus standard rodent chow and water were provided, and the diet was altered daily by replacing four of the foods with new items; hence, the animals did not receive the same foods for more than two consecutive days at a time. The body weights of the animals were measured between 11.00 and 12.00 weekly.

At 90 d of age, the estrous cycle was assessed by vaginal smear. The material was collected with the aid of a dropper containing 70% saline. The vaginal epithelium of the female rats was rinsed with this solution, and the collected material was immediately analyzed by optical microscopy to determine the current phase of the estrous cycle. Female rats in the pro-estrous phase with signs of sexual receptivity were mated with a control male rat. Mating was confirmed by the presence of sperm in a vaginal smear in the following morning, when possibly pregnant females from the two initial groups were subdivided into four groups: (1) a pre-gestation control chow diet group was kept on chow diet during pregnancy and lactation (CON–CON n = 12); (2) a pre-gestation control chow diet group placed on cafeteria diet from day 1 of pregnancy to the weaning of pups (CON–CAF n = 12); (3) a pre-gestation CAF group was kept on cafeteria diet during pregnancy and lactation (CAF–CAF n = 12) and (4) a pre-gestation CAF group placed on control chow diet from day 1 of pregnancy to the weaning of pups (CAF–CON n = 11). The experimental design is shown in Fig. 1. Approximately 5 days before delivery, pregnant females were individually housed and the presence of pups was checked twice a day. During pregnancy, females were weighed on alternate days. On the day of birth (Day 1), the number of pups was randomly culled into eight per dam to provide standardization of the litter size and to avoid differences in maternal care (mainly breastfeeding) between litters. Females whose litters had fewer than eight pups were excluded from the experiment. Shortly after standardization, the litters were weighed and remained without intervention until the 10th day when they were weighed again.

At weaning, 2 male pups from each litter were removed, weighed, and identified according to their maternal groups. Thereafter, the animals from each maternal group were randomly placed in acrylic boxes containing 5 animals each. Therefore, there were 4 boxes per group. The offspring of all groups received only water and standard rodent chow *ad libitum*. Food intake was monitored by weighing (g) the chow initially provided, subtracting the weight from the remaining amount. Total food consumption (from weaning to 90 d) was estimated for each animal by dividing total consumption of each box by 5 animals.

2.2. Male rat sexual behavior

At 90 d, 1 (virgin) male offspring of female rats from different experimental groups was submitted to the sexual behavior test, which occurred during the dark cycle between 7:00 p.m. and 10:00 p.m. Each rat was removed from the home cage and placed into a room contiguous to the animal laboratory, in an acrylic observation box (70 × 70 × 35 cm), being kept there for 15 min for habituation. Ten 3-month-old female Wistar rats were used to record male sexual behavior. The females were gonadectomized and induced to sexual receptivity by a sequential subcutaneous injection of 20 μg of estradiol (Ginestrol™, Köning, Argentina) 48 h, and 20 μg of estradiol plus 500 μg of progesterone (4-Pregnene-3,20-dione, Sigma, St Louis, USA) 6 h before testing. Sexual behavior was recorded with a digital camera for 20 min and the latency to the first mount, the frequency of mounts until the first intromission, and the latency to the first intromission and the frequency of intromissions were computed. Mount or intromission not observed within 20 min (frequency equal to zero) was computed as 1200 second latency [32,33].

Table 1
Food available to the animals on the cafeteria diet.

	Energy (kJ/100 g)	Carbohydrates (g/100 g)	Protein (g/100 g)	Fat (g/100 g)
Chow (NutriLab CR-1, Brazil)	1234	55	22	4
Salami (Majestade, Brazil)	1414	0	32	24
Bread Seven Boys (Seven Boys, Brazil)	1234	53	9	4
Snack Yokitos (Yoki, Brazil)	2008	60	6	24
Deliket Jelly Bean (Dori Alimentos, Brazil)	1590	95	0	0
Coca-Cola (Coca-Cola, Brazil)	178	11	0	0
Smoked sausage (Perdigão, Brazil)	1331	1	32	32
Chocolate cake (Nutrella, Brazil)	1360	50	12	12
Biscuit Maisena (Isabela, Brazil)	1799	73	12	12
Marshmallow (Fini, Brazil)	1423	80	0	0
Ham (Sadia, Brazil)	649	0	9	9
Snack Fritello (Pavioli, Brazil)	2125	52	29	29
Wafer Biscuit Chocolate (Bauducco, Brazil)	2176	63	27	27
Gumdrop Gomets (Dori Alimentos, Brazil)	1506	90	0	0

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